Letters

The real ethics of rationing

Purchasers, not surgeons, control waiting lists

EDITOR—I applaud the *BMJ*'s efforts to bring the important issue of rationing to the fore, but I cannot allow one aspect of Light's contribution to go without challenge.[1] Under the heading "parasitic privatisation" he says, "to top it off, the surgeons control the waiting lists" (to encourage patients into the private sector). I despair that someone of his academic stature should show such a lack of understanding of the purchaser-provider split that we have had in the NHS for the past six years. In the vast majority of cases we as surgeons have a contract with purchasers that places an upper limit on the number of patients on whom we can operate. Thus surgeons have no control over the number of patients having surgery and therefore, by definition, over the waiting list. The role of the surgeon is to prioritise, on clinical grounds, within the contracted number.

If surgeons are to operate on more patients then purchasers must contract for more operations. The onus to reduce waiting time, and therefore encourage patients not to use the private sector, lies solely with the purchasers.

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1 Light DW. The real ethics of rationing. *BMJ* 1997;315: 112-5. (12 July.)

Doctors should not be penalised for doing private work in spare time

EDITOR—Parts of Light's article on the ethics of rationing are an affront to all hard working consultants.[1] Of course the surgeon controls the waiting list to a certain extent—it is the surgeon who has assessed the patient who can decide on the urgency of the surgery needed. It is pernicious to imply that manipulation commonly takes place in order to direct patients towards the private sector. The problem is between the purchaser and the providing trust.

The organisational and financial restrictions on the NHS are the primary reason for the little time for which surgeons operate in the NHS. I would love to operate for 15 hours a week in the NHS as opposed to my present seven hours, but operating time is being reduced for financial reasons. I am distressed not to be able to get patients in sooner than three months. Yet Light has the effrontery to imply that this situation is in some way my fault.

Occasionally patients who have been seen in the NHS consult me privately. It would be detrimental to expose them to unnecessary radiation, not to mention a waste of time if radiographs have already been obtained. I therefore try to track down the previous radiographs and assure myself of the diagnosis. This is not stealing. What is at issue is what is best for that patient.

I work a great many hours in the NHS: the administrative side of the job makes for 12 hour days. I also, for a week at a time, provide emergency cover. It is not unreasonable to have a free half day during the week to pursue other activities. Junior doctors are entitled to a half day a week off. Whether I choose to study, prepare a lecture, play sport, or see private patients is up to me. The weekends are also my own time. I believe that the difference between full time and maximum part time contracts should be abolished. Why should I be penalised by losing part of my NHS salary for working privately in my spare time? The NHS gets highly trained professionals on the cheap. The starting salary for a consultant is £43 165, not £50 000 as stated in the article. Medical colleagues in the United States and Australia enjoy a much higher standard of living than I can aspire to.

Consultants' aim is to provide the best service that they can to people in need. They must not be held responsible for the inequities in health funding.

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1 Light DW. The real ethics of rationing. *BMJ* 1997;315: 112-5. (12 July.)

Conflict between private and national interests within NHS needs scrutinising

EDITOR—Light's article on the ethics of rationing points to a major source of inequity in the NHS.[1] Light could have further highlighted the scandal that not only may an NHS patient wait 3-6 months (or actually much longer) while a private patient waits 3-6 days but it may well be the same consultant providing both services. It must be wrong that a patient who feels forced to opt for private attention is attended by a consultant contracted on a whole time (or substantially so) basis to the NHS. Furthermore, this private service will often be provided in what for any ordinary employee would be regarded as the firm's time and even, sometimes, on the firm's premises. I cannot think of any other business or profession in which this would not be regarded as a clear conflict of interest. Most professions have their perks; medicine is no exception. But the question to be asked is whether their exercise competes in any way with the employer's business. No specious talk of income generation should obscure this.

Light could also have extended his argument to the use of capital. Luxurious private beds are now provided in NHS hospitals, while within a few minutes' walking distance terminally ill patients can lie for hours on trolleys, waiting in distress without pain relief or even having their thirst slaked or toilet needs attended to. Private funding initiatives can but compound this. The increasingly stark contrasts of wealth and poverty in Britain are being mirrored within our hospitals.

Surely the government needs to have a scrutiny into the conflict between private and national interests within the NHS. "New Labour" should remember what happened last time Labour was in power, when the then minister of health, Barbara Castle, sought to encourage whole time commitment but her proposals were resisted by those who engaged in part time practice. Her attempts to make whole time commitment more

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Letters will be edited and may be shortened.

attractive gave rise to protests from part time consultants who saw this as a gross deprivation for themselves. The current variant on this theme is the attempt to abolish what little distinction remains between whole timers and maximum part timers.

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Death rates from childhood leukaemia near nuclear sites

Numbers of observed deaths were closer to those expected after known risk factors were allowed for

EDITOR—Busby and Cato report a significant excess of deaths from childhood leukaemia during 1981-95 in the county districts of Newbury and south Oxfordshire and suggest that this might be linked to discharges of radionuclides from nuclear installations in the vicinity.[1] This study has two major limitations: the fact that survival from childhood leukaemia during the period studied was substantial[2] and the authors' failure to take into account any of the known risk factors for childhood leukaemia. In particular, the incidence of acute lymphoblastic leukaemia (which accounted for 64% of childhood deaths from leukaemia in the period considered) is higher in county districts that are rural and have a higher socioeconomic status and a higher level of child migration.[3 4]

Using the survival rates presented by Stiller,[2] we estimate that Busby and Cato are likely to have excluded from their study about two thirds of the cases that were diagnosed during 1981-95. While the incidence of leukaemia may reflect environmental factors, the death rate is likely to depend on genetic and clinical factors.[5]

Despite the difficulty of interpreting a study of childhood leukaemia based on deaths rather than incidence, we investigated the possible effect of known risk factors. Draper et al reported the incidence of childhood lymphocytic and unspecified leukaemia in county districts classified by urban or rural status and socioeconomic score.[3] We derived these indicators from 1981 census data for each of the county districts of interest and recalculated the expected number of deaths and the corresponding Poisson probability, allowing for these factors. We assumed that the risk of non-lymphocytic leukaemia and the survival of patients with leukaemia did not vary between county districts. We carried out a similar calculation using incidences of acute lymphoblastic leukaemia in county districts classified by level of child migration.[4]

The table shows that after allowance is made for these known risk factors, the numbers of deaths observed in Newbury and south Oxfordshire were much closer to those expected than were those given in the authors' letter. Census data show that south Oxfordshire had higher scores for both socioeconomic status and child migration in 1991 than 1981. Such demographic changes or the diversity of origin of migrants, which is also strongly associated with an increased risk of childhood acute lymphoblastic leukaemia,[4] might explain most of the increased death rate.

This study was funded by the United Kingdom Coordinating Committee on Cancer Research, Westlakes Research Institute, and North of England Children's Cancer Research Fund. British Nuclear Fuels plc is a major client of Westlakes Research Institute, but neither the company nor the institute was involved in any aspect of the design, analysis, or reporting of the study.

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1 Busby C, Cato MS. Death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire. *BMJ* 1997;315:309. (2 August.)
2 Stiller CA. Population based survival rates for childhood cancer in Britain, 1980-91. *BMJ* 1994;309:1612-6.
3 Draper GJ, Vincent TJ, O'Connor CM, Stiller CA. Socio-economic factors and variation in incidence rates between county districts. In: Draper G, ed. *The geographical epidemiology of childhood leukaemia and non Hodgkin lymphomas in Great Britain 1966-83*. London: HMSO, 1991:37-46.
4 Stiller CA, Boyle PJ. Effect of population mixing and socioeconomic status in England and Wales, 1979-85, on lymphoblastic leukaemia in children. *BMJ* 1996;313: 1297-300.
5 Ross JA, Davies SM, Potter JD, Robison LL. Epidemiology of childhood leukaemia, with a focus on infants. *Epidemiol Rev* 1994;16:243-72.

Other studies showed that radiation levels in Newbury area were low

EDITOR—We believe that Busby and Cato have been selective in their use of data to show that death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire.[1] Their interpretation of the data is both subjective and unsubstantiated.

They refer to studies reported by the Commission on Medical Aspects of Radiation in the Environment[2] but fail to mention that this report concluded: "in our judgement, the authorised and accidental radioactive discharges from AWRE [Atomic Weapons Research Establishment] Aldermaston, ROF [Royal Ordnance Factory] Burghfield and AERE [Atomic Energy Research Establishment] Harwell are far too low to account for the observed increase in childhood cancer incidence in the area."[2]

Two independent radiation surveys of the area have recently been carried out as a result of the concerns over the incidence of childhood leukaemia in south Newbury.[3 4] These showed that the levels in the Newbury area generally were low by British standards and certainly far too low for there to be any possibility that they might cause observable increases in rates of cancer. To date, no studies have suggested that current risk estimates for internal exposures at low doses are underestimated by the many orders of magnitude necessary to account for the additional cases of leukaemia.

We believe that our activities at AWE are extremely unlikely to be the cause of the increased incidence of leukaemia or of other cancers. However, we are not complacent: we are involved with Newbury District Council's current initiative to find the cause, and we will continue to cooperate with the Commission on Medical Aspects of Radiation in the Environment and other eminent bodies in their efforts to establish the facts.

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1 Busby C, Cato MS. Death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire. *BMJ* 1997;315:309. (2 August.)
2 Commission on Medical Aspects of Radiation in the Environment. *Third report. Report on the incidence of childhood cancer in the west Berkshire and north Hampshire area.* London: HMSO, 1989.

Estimation of expected numbers of deaths from leukaemia in children aged 0-14 years in county districts in Berkshire and Oxfordshire, allowing for known risk factors

	Type of county district				Calculations using relative risks from:					
	Indicators used by Draper et al[3]		Indicator used by Stiller and Boyle[4]		Draper et al[3]			Stiller and Boyle[4]		
County district	Urban/ rural	Socioeconomic score*	Child migration	Observed deaths (O)	Expected deaths (E)	Relative risk (O/E)	Poisson P	Expected deaths (E)	Relative risk (O/E)	Poisson P
Oxford	Urban	4	Medium	3	3.7	0.77	0.72	3.9	0.74	0.75
Cherwell	Rural	2	High	7	5.5	1.18	0.31	5.4	1.18	0.29
West Oxford	Rural	2	High	5	3.7	1.25	0.31	3.6	1.25	0.30
South Oxfordshire	Rural	2	Medium	12	5.0	2.20	0.01	5.0	2.33	0.01
Vale of the White Horse	Rural	1	Medium	3	5.0	0.59	0.87	4.5	0.65	0.82
Newbury	Rural	1	Medium	11	6.4	1.67	0.06	5.8	1.84	0.03
Reading	Urban	4	Medium	6	5.0	1.16	0.39	5.2	1.11	0.42

*Fifth: 1=high, 5=low.

3 Croudace W, Sanderson DCW, Warwick PE, Allyson JD. *A regional study of the radiation environment of Greenham Common, Newbury district and the surrounding area.* Newbury: Newbury District Council, 1997.
4 Fry FA, Wilkins BT. *An assessment of radionuclide levels around the former air base at Greenham Common, Berkshire.* Didcot: National Radiological Protection Board, 1996. (NRPB Report M752.)

Epidemiological studies must define their cohort objectively

EDITOR—Busby and Cato report that death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire.[1] Their flawed methodology might be excused if their data uncovered an interesting finding in some poorly researched topic. But the incidence of childhood leukaemia in relation to nuclear installations hardly comes into that category.[2]

Childhood leukaemias show considerable geographical and temporal variations in incidence, and an infective agent may well be important in their aetiology.[3] It would be easy enough to find combinations of time and place in which any random environmental agent showed a correlation with the disease. That is why epidemiological studies should be scrupulous about defining their cohort objectively. Busby and Cato, however, have chosen a curious area with the nuclear sites of Aldermaston, Burghfield, and Harwell clustered at its southern end. It includes all the districts of Oxfordshire but excludes districts in Berkshire in close proximity to Burghfield and Aldermaston. There seems to be no rationale for choosing the study area. Similar arguments apply to the authors' choice of time period.

A second strong requirement for a professional study is that some objective measure of exposure is provided. On radioactive contamination, Busby and Cato refer readers to the 1989 report of the Committee on Medical Aspects of Radiation in the Environment[4] and assure us that "data in the committee's report suggest that south Oxfordshire would be most strongly affected followed by Newbury." Readers who look for such a ranking of county districts in the report will be disappointed. The report provides plenty of data, but to extrapolate from these to average exposure in the relevant districts would require much more information, which is not provided. It is also worth noting that one of the main conclusions of the report was: "In our judgement, the authorised and accidental radioactive discharges from AWRE [Atomic Weapons Research Establishment] Aldermaston, ROF [Royal Ordnance Factory] Burghfield and AERE [Atomic Energy Research Establishment] Harwell are far too low to account for the observed increase in childhood cancer incidence in the area."

Fortunately, the serious issue of childhood leukaemia around these nuclear sites has been studied with an objective methodology. Bithell et al found no evidence of a raised incidence of childhood leukaemia within a 25 km radius of any of these sites.[5] In the cases of Aldermaston and Harwell they found no trend of decreasing incidence with distance from the site, and, though there was a trend for Burghfield, they concluded that emissions were unlikely to be the cause.

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1 Busby C, Cato MS. Death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire. *BMJ* 1997; 315:309. (2 August.)
2 Beral V, Roman E, Bobrow M. *Childhood cancer and nuclear installations.* London: BMJ Publishing Group, 1993.
3 Kinlen LJ. Epidemiological evidence for an infective basis in childhood leukaemia. *Br J Cancer* 1995;71:1-52.
4 Committee on Medical Aspects of Radiation in the Environment. *Third report. Report on the incidence of childhood cancer in the west Berkshire and north Hampshire area, in which are situated the Atomic Weapons Research Establishment, Aldermaston and the Royal Ordnance Factory, Burghfield.* London: HMSO, 1989.
5 Bithell JF, Dutton SJ, Draper GJ, Neary NM. The distribution of childhood leukaemias and non-Hodgkin's lymphomas near nuclear installations in England and Wales. *BMJ* 1994;309:501-5.

Findings were probably due to chance fluctuations in small numbers of deaths

EDITOR—Busby and Cato analysed childhood mortality from leukaemia for seven county districts in the area of Oxfordshire and Berkshire containing three nuclear sites and found increased rates for south Oxfordshire and Newbury.[1] They suggested that these districts would have been the two most affected by radioactive contamination.

We have three comments on their analysis. Firstly, the contamination seems to be insufficient to cause the increased rate of leukaemia, though they argue that the carcinogenic risk of ionising radiation has been underestimated. Secondly, they do not adequately explain their reasons for suggesting that children in south Oxfordshire and Newbury would be the most contaminated. Thirdly, as shown below, the incidence rates in these two districts are not unusually high when compared with those in some adjacent counties.

We used the National Registry of Childhood Tumours[2] to calculate an age standardised incidence rates of leukaemia at ages 0-14 years for each county district of England, Wales, and Scotland during 1969-93. This is a uniformly weighted average of the rates at ages 0-4, 5-9, and 10-14 years.

The table shows the numbers of cases and standardised incidence rates for the county districts analysed by Busby and Cato, and the ratios of these rates to that for England and Wales, for comparison with their mortality ratios. One would expect these ratios to be similar within each county district, but there are major differences. The analysis based on incidence should be more correct than that based on mortality, since the numbers are larger, and mortality rates could be influenced by factors other than aetiological ones. The most plausible explanation for the differences, however, seems to be that they are due to chance fluctuations in the small numbers of deaths occurring. Whatever the explanation, the main finding is that the more reliable analysis based on incidence does not support the suggestion that there are major differences in childhood leukaemia rates between these county districts.

In a previous analysis the rates of childhood leukaemia were higher than average for some counties in this area.[3] The rates for Berkshire, Buckinghamshire, Hampshire, Oxfordshire, and Wiltshire for 1969-93 were 44.0, 49.0, 36.9, 38.4, and 47.0 per million; that for England, Wales, and Scotland combined was 38.3. Such higher rates seem to be associated with higher socioeconomic status, though this may be another aspect of the population mixing effect.[4 5] An analysis of these data in relation to socioeconomic status and to proximity to nuclear installations is in preparation.

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1 Busby C, Cato MS. Death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire. *BMJ* 1997;315:309. (2 August.)
2 Stiller CA, Allen MB, Eatock EM. Childhood cancer in Britain; the national registry of childhood tumours and incidence rates 1978-1987. *Eur J Cancer* 1995;31A:2028-34.
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Rate of childhood leukaemia in county districts in vicinity of nuclear installations in Oxfordshire and Berkshire (registrations 1969-93, National Registry of Childhood Tumours)

	Average annual population aged 0-14 (1969-93)	No of cases	Age standardised rate*	Rank†	Ratio of rates to those for England and Wales	
					Incidence‡	Mortality§
Oxford city	19 559	17	35.7	276	0.93	0.78
Cherwell	25 733	24	37.3	239	0.97	1.30
West Oxfordshire	18 587	25	53.7	42	1.40	1.40
South Oxfordshire	28 256	32	46.1	101	1.20	2.45
Vale of White Horse	22 353	11	20.1	439	0.52	0.68
Newbury	27 695	34	49.2	69	1.28	1.93
Reading	28 446	30	41.8	162	1.09	1.15
England and Wales	10 407 413	9865	38.3	—	1.00	1.00

*Annual registration rate per million children that would be observed in population with equal numbers of children in age groups 0-4, 5-9, and 10-14 if age specific rates were those in specified area.
†Age standardised rates are ranked for all 459 county districts in England, Wales, and Scotland. Rank 1 is highest.
‡Age standardised rate relative to that for England and Wales.
§Ratio of observed to expected deaths from Busby and Cato.[1]

Place of residence at diagnosis and at death may be different

EDITOR—As the consultant haematologist who has been responsible for the care of most of the children with leukaemia in west Berkshire for the past 25 years, I would like to raise some words of caution about Busby and Cato's interpretation of death rates from leukaemia in Berkshire.[1] Codes 204-208 in the *International Classification of Diseases* encompass diseases with very different biological behaviour. Some of these diseases, such as chronic myeloid leukaemia and acute myeloid leukaemia, are both rare and difficult to treat; if, by chance, several children with these diagnoses present in a short period, they can skew the picture.

The cure rate for acute lymphoblastic leukaemia, the commonest form of childhood leukaemia, has almost doubled in the period under discussion. This is also likely to cause discrepancies in the numbers of deaths if a handful of children present late in their disease or with adverse biological features in a short period.

Children with acute lymphoblastic leukaemia in west Berkshire have been entered into the Medical Research Council's acute lymphoblastic leukaemia trials since 1972. Over this longer period there has been no increase in relapse or death rates among these children compared with the other children in the trial (S Richards, personal communication).

Also of note is the fact that treatment may now prolong life for many years in those who cannot be cured. Thus leukaemia may be diagnosed while children are living in areas far removed from their residence at death. It is therefore more rational to look at the incidence of childhood leukaemia for epidemiological purposes.

The incidence of childhood leukaemia is indeed raised in west Berkshire,[3] and work is being undertaken to assess whether this is related in any way to environmental factors. Previous work has shown a doubling of the risk of leukaemia in young children (aged 0-4 years) living close to nuclear sites.[4] In view of the concerns raised by Busby and Cato and the continuing raised incidence of the disease, this needs to be reassessed, preferably by scientists independent of the nuclear industry or environmental pressure groups, so that the local population may have confidence in their results.

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1 Busby C, Cato MS. Death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire. *BMJ* 1997;315:309. (2 August.)
2 Barton C, Ryder H. Leukaemia near La Hague nuclear plant. *BMJ* 1997;314:1554. (24 May.)
3 Roman E, Beral V, Carpenter L, Watson A, Barton C, Ryder H, et al. Childhood leukaemia in West Berkshire and Basingstoke and North Hampshire District Health Authorities in relation to nuclear establishments in the vicinity. *BMJ* 1987;294:597-602.

Authors' reply

EDITOR—We are criticised for using mortality rather than incidence data. Although aware of their limitations, we were forced to use mortality data because of the refusal by the Oxford Cancer Intelligence Unit to release small area incidence data, despite repeated applications and pressure from local elected councillors and members of the public. We have now obtained permission from Berkshire Ethical Committee to be given figures for Newbury, but South Oxfordshire Ethical Committee continues to refuse to release its figures. This is of concern to us since the director of Oxfordshire Health Authority was previously director of the Harwell and Culham laboratories, which may suggest to the public a conflict of interest.

Dickinson et al refine our study by applying known risk factors to calculate a slightly higher expected number of deaths than we found. They essentially confirm our conclusions although reduce the relative risk by a small amount (5-10%).

The strong letters from the nuclear establishments were expected, though they are milder than the recent review in their house magazine, *Nuclear Energy*, which accused us of sadism. On the matter of risk factor errors we refer these authors to Rooney et al's study of prostatic cancer in employees of nuclear establishments who were internally contaminated with radionuclides,[1] and Atkinson et al's response,[2] and to the recent report by Jeffreys et al showing a doubling in minisatellite DNA mutation in children exposed to low level radioactive contamination from Chernobyl.[3] This latter report exposes an error of over 5000-fold in the perception of risks of radiogenic mutation based on experience since the bombing of Hiroshima.

Draper and Vincent compare our figures based on mortality for 1981-95 with figures based on published incidence data. The precise period that they used for their incidence data is unclear: their letter says 1969-93, but the reference that they cite is for 1978-87, which misses almost half the period that we covered. Given the increase in the incidence of leukemia in children aged 0-4 that occurred in England and Wales after 1987 (data from the Office of Population Censuses and Surveys[4] and Wales Cancer Registry), the relevance of this exercise is uncertain.

Barton argues for independent research. No one is independent. Who pays the researchers? Whose prospects for advancement would survive being the messenger of the bad news that the environment is being poisoned? Attempts to play down real environmental effects are much more popular.[5] In our case, no one pays: we do this work, on a shoestring, because we believe that we have to, for our children and everyone else's.

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1 Rooney C, Maconochie N, Fraser P, Davies G, Beral V. Case-control study of prostatic cancer in employees of the United Kingdom Atomic Energy Authority. *BMJ* 1993; 307:1391-7.
2 Atkinson WD, Marshall M, Wade BO. Prostatic cancer and radionuclides. *BMJ* 1993;308:268.
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4 Office of Population Censuses and Surveys. *Cancer statistics: registrations: cases of diagnosed cancer registered in England and Wales.* London: OPCS. (Series MBI.)
5 Olsen FO, Martuzzi M, Elliott P. Cluster analysis and disease mapping—why, when, and how? A step by step guide. *BMJ* 1996;313:863-6.

Number of deaths in Newbury area is not increased

EDITOR—Busby and Cato's letter saying that death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire gives an incomplete picture of mortality in the area served by Newbury District Council.[1] After concerns were raised about leukaemia in the area, deaths from this cause (*International Classification of Diseases* codes 204-8) in the area served by Newbury council (population roughly 140 000) were examined for the period 1980-95. Actual numbers of deaths were compared with expected numbers using figures for the population of and deaths in England and Wales over the same period.

Observed and expected numbers of deaths from leukaemia by age group in area covered by Newbury District Council, 1980-95

Age group (years)	Observed No	Expected No	Mortality ratio
0-24	14	11.7	1.20
0-4	3	1.92	1.56
5-14	9	4.51	2.00
15-24	2	5.27	0.38
25-64	38	43.3	0.88
⩾65	71	80.5	0.88
Total	123	135.5	0.91

Result for age group 5-14 was significant (P<0.05) with one sided test but not with two sided test.

The table shows the number of deaths from leukaemia in the age bands 0-24, 25-64, and ⩾65. In no age band was the number of deaths significantly different from that expected. Indeed, overall the number was below that expected. The table also shows deaths from leukaemia for the age bands 0-4, 5-14, and 15-24; in this case there was a significantly increased number of deaths in the age group 5-14 when a one sided Poisson distribution was used.

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1 Busby C, Cato MS. Death rates from leukaemia are higher than expected in areas around nuclear sites in Berkshire and Oxfordshire. *BMJ* 1997;315:309. (2 August.)

In screening for breast cancer, clinical examination is as effective as mammography

EDITOR—Melvin J Silverstein betrays his personal bias when he states that mammography is the best diagnostic tool for early

detection of breast cancer.[1] To prove his point he compares 10 year survival in patients with non-palpable invasive breast cancers <1 cm detected at screening with that in patients with palpable cancers seen in hospital clinics, and he concludes that the survival of the former group is 15-40% better than that of the latter.

The truth is that, in the setting of breast cancer screening, a careful clinical examination is as effective as mammography.[2] Although the real usefulness of mammography lies in the detection of non-palpable cancers, most cancers detected at mammographic screening are palpable and there is no evidence to suggest that the detection of non-palpable cancers has any impact on reducing mortality.[3] A Canadian study of women aged 50-59 in which physical examination was compared with physical examination plus mammography found no added benefit for the mammography arm even after a follow up of 10 years (A B Miller, personal communication).

It is not difficult to understand why so much collective energy has been expended to discredit the Canadian study when there is published evidence to show that the cancer detection rates and the incidence of interval cancers in that study were identical with those in the largest and the most classical screening study of the same era—the breast cancer detection demonstration project.[4] Until the detection of non-palpable cancers is shown to reduce the death rate from breast cancer (even among women over 50), rather than merely improve survival, the claim that this technique is the best diagnostic tool will remain scientifically untenable.

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1 Silverstein MJ. Diagnosis and treatment of early breast cancer. *BMJ* 1997;314:1736-9. (14 June.)
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Not all self help groups discourage sick doctors from being members

EDITOR—I am glad to say that my encounters with the Manic-Depressive Fellowship have been the reverse of N J Stafford's experience with a self help group[1]: I have been warmly welcomed. I am currently organising the fellowship's research papers and providing short abstracts of the medical literature on manic depression. I have not come across the prejudice that Stafford describes and am viewed as an asset because of my medical background.

I am also involved with the Doctors' Support Network.[2] This is a self help organisation for doctors who have had significant mental health problems. The focus is meeting with other doctors who have had similar problems, in an environment where it feels safe to talk about these experiences. This group may be contacted on 0171 727 3738 or through PO Box 12826, London SW4 8ZL.

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Incidence of gastrointestinal side effects due to alendronate is high in clinical practice

EDITOR—Recent advertisements in the *BMJ* for alendronate sodium (Fosamax), which is used in osteoporosis, have stated that "side effects, which usually were mild, generally did not require discontinuation of therapy." The overall incidence of upper gastrointestinal side effects in clinical trials is given as 1%. Our experience of alendronate in clinical practice is different.

We have studied 77 consecutive women (mean age 65 (range 27-83)) who were treated with alendronate 10 mg daily for nine to 66 weeks. All were given instructions about how to take the drug, which were reinforced after March 1996 by the advice that they should not lie down after taking it. They were questioned in detail about compliance with the instructions and side effects.

Twenty four (31%) had side effects. Two of these developed rashes, which resolved when they stopped the drug. The other 22 had mainly upper gastrointestinal problems, particularly problems suggesting gastro-oesophageal reflux: dyspepsia (16 patients), heartburn (14), retrosternal pain (9), dysphagia (5), nausea (8), and vomiting (3). Many had more than one symptom. One patient developed an oesophageal stricture, which required dilatation. Twenty stopped taking alendronate because of the severity of their symptoms.

Problems developed between two and 50 weeks (mean 20) after the start of treatment. Sixteen lay down after swallowing alendronate, of whom seven developed side effects. Twenty one had a history of upper gastrointestinal disease, of whom six developed side effects, but two had also lain down after taking the drug.

The company,[1] and others,[2 3] have suggested that rigorous adherence to instructions minimises the risk of oesophagitis. Our experience is that an appreciable number of patients will still be intolerant of alendronate.

One explanation for the large difference in the incidence of side effects may be that patients taking regular drug treatment for dyspepsia were excluded from the alendronate fracture intervention trial.[4] The incidence of gastrointestinal side effects that we found is similar to that reported with oral pamidronate,[5] an aminobisphosphonate with a similar structure to alendronate. We believe that patients taking alendronate need to be monitored regularly, long term, for compliance and side effects.

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Communication between GPs and cooperatives is poor for terminally ill patients

EDITOR—In a study of a general practice cooperative, Salisbury pointed out that pressure for change in out of hours care has come almost entirely from the medical profession and that it is important to consider the patients' perspective.[1] Our work has identified that continuity and familiarity of professional carers are particularly important to terminally ill patients in the community.[2 3]

By looking at medical records, we undertook a retrospective audit of palliative care in the Cambridge general practice cooperative, which covers 210 000 patients. During August 1996 there were 2202 patient contacts, 53 of which were for 40 patients recognisably in the terminal phase of their illness. Forty three visits (including 10 to certify expected death) and 10 telephone consultations were made.

Three areas of discontinuity of care were identified. Firstly, none of these patients' general practitioners had handed over any information to the cooperative. In each case the cooperative doctor was managing patients close to death, or meeting newly bereaved relatives, without any knowledge of the physical, psychological, and social backgrounds. Secondly, continuity of care within the cooperative was lacking. Six patients had two contacts with the cooperative, two had three, and one had four (13 repeat contacts altogether); only one patient saw the same doctor twice. For nine of these 13 repeat contacts, the cooperative doctor was unaware of information from previous contacts with the cooperative. Thirdly, rapid handover from the cooperative to the general practitioner was patchy. While details of all deaths were routinely faxed to the general practitioner the next working day, this occurred for only half of the telephone contacts and visits for dying patients; for the remainder, the general practitioner was informed by post.

The loss of personal continuity of care may be mitigated by the general practitioner handing over information concerning vulnerable patients to the cooperative, by ensuring the communication of relevant information within the cooperative, and by routinely faxing details concerning dying patients to general practitioners; it is planned to introduce these changes locally. Currently available computer programs could readily facilitate such communication, thereby maximising continuity.

The Cambridge cooperative has established a good reputation overall but seems not to function well with terminally ill patients, who constituted a small but important part of its workload. If primary care is to remain central to palliative and terminal care,[4] every effort must be made to minimise the adverse impact of the recent changes in out of hours care.

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Geriatric depression scale can be used in older people in primary care

Editor—In their review of common mental health problems in primary care, Craig and Boardman make no mention of the problems of detecting and managing such problems in older people.[1] Considerable evidence from community and primary care based studies, including our own, shows that depression in older people is both common and associated with high consultation rates in general practice but seldom receives appropriate treatment.[2 3] We have also shown that older people are less likely than their younger counterparts to be willing to seek any form of help (informal or professional) for depression.[4]

We agree with Craig and Boardman that a positive attitude to mental disorder and appropriate interview skills are important in increasing detection rates, though this has not been shown formally in older patients. We suggest, however, that the potential utility of screening questionnaires should also be considered. We have shown, for example, that the geriatric depression scale, a simple 15 item questionnaire recommended by the Royal College of General Practitioners, is highly acceptable in primary care practice as well as having excellent specificity and sensitivity in detecting depressive illness.[3 5] As few as four questions derived from the geriatric depression scale (table) can detect most cases of depression in older people in primary care.[4]

Four item version of geriatric depression scale (adapted from D'Ath et al[3])

Question	Answer*
Are you basically satisfied with your life?	Yes/NO
Do you feel that your life is empty?	YES/No
Are you afraid that something bad is going to happen to you?	YES/No
Do you feel happy most of the time?	Yes/NO

*Answers in capitals score 1 point. Score 0: probably not depressed; score ⩾2: probably depressed.

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1 Craig TKJ, Boardman AP. ABC of mental health: Common mental health problems in primary care. *BMJ* 1997;314:1609-12. (31 May.)
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Is the clinical course of HIV infection changing?

Finding is disheartening

Editor—Sinicco et al reported a worse prognosis for patients who became HIV positive between 1991 and 1995 compared with those who seroconverted before 1991.[1] Their findings are unexpected, and caution must be exercised to avoid overinterpreting them in the light of results from several studies, including that by the UK Register of HIV Seroconverters (XI international conference on AIDS, Vancouver, 1996), which have not detected a change in the time from seroconversion to AIDS or death. Indeed, a slight improvement in prognosis was reported in one study from the Netherlands of homosexual men who seroconverted between 1985 and 1993.[2] Encouraging results from clinical trials suggest that the use of dual antiretroviral treatment, which began in the mid-1990s, is likely to prolong survival[3 4]; depending on the rate of uptake, this would be expected to lead to a better prognosis in the more recent time periods. Further improvements are anticipated with increasing use of drug regimens that include two nucleoside analogues and a protease inhibitor.

Although data on CD4 cell count, which were much used by Sinicco et al, are useful markers of progression of disease, they are highly dependent on the frequency of clinical follow up and of the collection of specimens and may be increased temporarily by antiretroviral drugs. Time to death and AIDS are end points independent of the protocol for monitoring the CD4 cell count and bear the most relevance to patients with HIV infection.

Methodological and statistical issues that influence the eligibility criteria for inclusion in a study, such as frequency of attendance and follow up, changes in the rate of loss to follow up over time, as well as the possibility of preferentially including patients whose disease progressed rapidly in the later time period may wholly or partly explain why Sinicco et al's observations differ from those made by other investigators conducting similar studies. Their finding is, nevertheless, disheartening at a time of optimism and serves to highlight the need to continue to monitor the incubation period to AIDS and survival from HIV seroconversion on a national and international level.

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1 Sinicco A, Fora R, Raiteri R, Sciandra M, Bechis G, Calvo MM, et al. Is the clinical course of HIV-1 changing? Cohort study. *BMJ* 1997;314:1232-7. (26 April.)
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Exclusion of people not followed up for 12 months may have biased results

Editor—Sinicco et al's paper reports a temporal trend to faster progression of disease and more rapid decline in CD4 cell count in patients infected with HIV in more recent years.[1] We were surprised by this, particularly as these results are at odds with those of similar studies which did not find any evidence of a trend.[2 3] One possible explanation for this apparent trend that Sinicco et al did not consider is the criteria they applied for including patients in their analysis. A total of 4134 patients were tested for HIV antibody between September 1985 and January 1996. Patients were included in the analysis if they had a positive result of an HIV test preceded by a negative result within 12 months, a measurement of their CD4 cell count within six months of their positive HIV test result, and at least 12 months' follow up after entry (presumably the estimated date of seroconversion).

Because patients infected with HIV often attend clinics only when their immune system is compromised and they begin to experience symptoms, the last of these criteria may have selectively excluded patients with a slower progression of disease and

decline in CD4 cell counts from the group of patients who seroconverted more recently.

So that readers can assess the extent of any bias introduced by these inclusion criteria, the authors could tabulate, in each group defined by time of infection, the number of patients who had a positive HIV test result preceded within 12 months by a negative result but who were excluded from the analysis because they failed to satisfy the 12 month follow up criterion. If there were an appreciable number of such patients, and if their proportion increased in the groups with more recent infection, this would suggest that the apparent temporal trend to faster progression of disease could be due to a bias introduced by the inclusion criteria. The magnitude of any bias could be assessed by further statistical analyses—for example, including as AIDS free to the end of 1995 all patients with a positive HIV test result preceded within 12 months by a negative result but who were previously excluded due to little follow up.

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1 Sinicco A, Fora R, Raiteri R, Sciandra M, Bechis G, Calvo MM, et al. Is the clinical course of HIV-1 changing? Cohort study. *BMJ* 1997;314:1232-7. (26 April.)
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Study's censoring strategy may be source of bias

EDITOR—Sinicco et al report that patients who were infected with HIV after December 1989 progress faster to certain CD4 cell counts than do patients infected during the 1980s.[1] This seems to support the speculation that the strains that have infected the population in recent years have been more virulent. If this is true then patients who seroconverted recently may need to be treated earlier than is currently advised. Although cohort studies of patients with known dates of HIV infection are the best source of data for evaluating temporal change in the incubation period at a population level, they may be prone to bias. The censoring strategy may be a major and not obvious source of bias. Criteria for inclusion in Sinicco et al's analysis were one year of follow up (that is, seroconversion happened at least one year before the closing date of the analysis, which was in January 1996) and having a measurement of the CD4 cell count at enrolment. The time taken to reach certain CD4 cell counts (500, 400, 200×10^6 cells/l) was the end point. Assuming that patients' follow up was censored at the date of their last measurement of the CD4 cell count, a given patient's follow up might be censored differently if he or she seroconverted early in the epidemic or shortly before the closing date of the study. For example, a person whose last CD4 cell count was above the chosen CD4 cell count end point two years after enrolment would be censored on the date of this measurement if he or she seroconverted early in the epidemic but would be censored at enrolment (that is, the date of the last CD4 cell count) and so excluded from the analysis if he or she seroconverted one year before the closing date of the study (those who seroconverted in January 1995). Thus the analyses may be biased by the exclusion of patients whose disease is likely to have progressed slowly from the group who had seroconverted recently. It would be interesting to see results from analyses that only the first year of follow up for all the patients, ignoring the visits that occurred after this. In this way the same censoring strategy would have been applied to patients seroconverting in different years.

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Authors' reply

EDITOR—We did not neglect the encouraging data from clinical trials of prolonged survival in HIV positive patients treated with early combination treatment. However, we did not consider treatment in our analysis, and the patients started treatment at the same CD4 cell count after we observed more rapid loss of CD4 cells within one year of seroconversion in those who serocoverted after December 1989. Indeed, we underlined the importance of timing treatment with the new drugs for better prognosis. Monotherapy could select more virulent strains and be responsible for faster progression.[1 2] These more aggressive strains might spread rapidly in urban areas. We did not consider survival as an end point, but further analysis after the introduction of combination treatment showed that survival of the different seroconversion groups tended to be similar, so it remains difficult to evaluate the effect of the new treatments because of the short follow up. In our opinion, the choice of AIDS defining events as end points may be as prone as the CD4 cell count to misjudgment, if not more so. In fact, the diagnosis of AIDS usually depends on more factors than the CD4 cell count. We cannot exclude statistical bias, but attempts were made to control the frequency of attendance and rate of loss to follow up. These features were similar in the different groups. We did not observe any difference in the loss to follow up in particular groups of patients, and we did not select patients with a poor prognosis, because follow up visits were scheduled and independent of clinical status. The inclusion criterion of 12 months' follow up applied only to the patients who seroconverted in 1992-5, to avoid the effects of short follow up. In the subsequent analysis, however, a decrease in the CD4 cell count in the patients in this group who were excluded because of less than one year's follow up was similar to that in the patients who were included. As for censoring strategy, we agree with Cozzi Lepri et al about the potential censoring of patients whose disease progressed slowly in the group who had seroconverted recently. Subsequent analysis confirmed a faster decline in the CD4 cell count in those who had seroconverted recently, at least until the start of the new therapeutic protocols. Finally, the suggestion that only the first year of follow up should be considered implies a reliance primarily on the viral load, which is information that we had for only some of the patients.

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Home grown asparagus is cheap

EDITOR—Macrae and Davies berate the Health Education Board for Scotland on economic grounds for the fact that the cover of its booklet *Eating for Health* features an asparagus spear.[1] A packet of asparagus seed costs 85p. My asparagus bed produces about 10 kg per season, and a bed should crop for 20 years. It thrives on compost from kitchen waste and wood ash (both free), and apart from manual removal of asparagus beetle, autumn clearing, and occasional weeding it requires no other attention. This works out at 0.43p/kg and offers far better value for money than the £2.03/kg for the ginger biscuits that the authors recommend.

I appreciate that not everyone has the space to grow vegetables, but most towns offer allotments to rent, many of which remain unrented and overgrown.

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1 Macrae WA, Davies HTO. Let them eat asparagus. *BMJ* 1997;315:124. (12 July.)